
A glucagon-like endocrine pathway in *Drosophila* modulates both lipid and carbohydrate homeostasis.

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Public Summary:

The regulation of energy homeostasis is fundamental to all organisms. The *Drosophila* fat body serves as a repository for both triglycerides and glycogen, combining the energy storage functions of mammalian adipose and hepatic tissues, respectively. Here we show that mutation of the *Drosophila* adipokinetic hormone receptor (AKHR), a functional analog of the mammalian glucagon receptor, leads to abnormal accumulation of both lipid and carbohydrate. As a consequence of their obese phenotypes, AKHR mutants are markedly starvation resistant. We show that AKHR is expressed in the fat body, and, intriguingly, in a subset of gustatory neurons that mediate sweet taste. Genetic rescue experiments establish that the metabolic phenotypes arise exclusively from the fat body AKHR expression. Behavioral experiments demonstrate that AKHR mutants are neither sedentary nor hyperphagic, suggesting the metabolic abnormalities derive from a genetic propensity to retain energy stores. Taken together, our results indicate that a single endocrine pathway contributes to both lipid and carbohydrate catabolism in the *Drosophila* fat body.

Scientific Abstract:

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